Original Article

Sclerosing peritonitis: the experience in Australia

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Abstract

Background. Sclerosing peritonitis (SP) is a rare but serious complication of peritoneal dialysis (PD). Small-bowel obstruction (SBO) due to encapsulation, dense adhesions, or mural fibrous is characteristic, often associated with peritonitis. The aim of the study was to determine the incidence, clinical features, effect of duration of dialysis, and other possible aetiological factors in severe SP.

Methods. All dialysis units in Australia were surveyed for possible cases up to 1994. Patients were included if there was either surgical or radiological evidence of sclerosing encapsulating peritonitis or SBO with tanned or thickened peritoneum in the absence of other causes of SBO.

Results. Fifty-four patients were analysed. The duration of continuous PD was mean 52 ± 30 months, median 48 months and range 8–127 months. Nineteen cases were diagnosed between 1980 and 1989 and 35 between 1990 and 1994, giving mean annual incidences 1.9 and 4.2 per 1000 PD periods respectively. The overall prevalence was 0.7%, which increased progressively with the duration of PD being 1.9, 6.4, 10.8, and 19.4% for patients on dialysis for >2, 5, 6 and 8 years repectively. Sclerosing encapsulating peritonitis was diagnosed in 87% of cases, SBO in 92%, and haemo-peritoneum in 8%. Peritoneal calcification was present in seven cases, all of which had been on PD >7 years. Peritonitis was associated with 38% of cases with fungal infection in 7%. Treatment with immunosup-pression in five patients appeared to result in a favour-able outcome in three. The mortality rate was 56%.

Conclusion. Severe sclerosing peritonitis is a serious complication of peritoneal dialysis and there is a time dependent increase on CAPD.

Key words: peritoneal dialysis; sclerosing peritonitis

Introduction

Sclerosing peritonitis is an inflammatory process affecting the peritoneum diffusely, transforming it into

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fibrous tissue. There is a spectrum of changes ranging from opacification, through tanned or thickened peri-toneum, to sclerosing encapsulating peritonitis that encapsulates the small bowel, leading to bowel obstruc-tion through obliteration of the peritoneal cavity or through adhesions. Small-bowel obstruction may also occur because of mural fibrosis in which the fibrotic process invades the outer wall of the small bowel. With mural fibrosis the peritoneum is usually tanned without encapsulation or adhesions [1].

Opacification of the peritoneum is due to changes in the submesothelial tissue with disorganization of the collagen fibres and expansion of matrix ground sub-stance. 'Tanned' peritoneum is a distinctive condition where the peritoneum is dry, wrinkled, and light brown in colour with a leathery appearance. Microscopically the outer portion of the peritoneum has been replaced by an acellular band of hyalinized collagen. The meso-thelium is absent. Progression of this process inwards results in fibrosis of the outer wall of the small bowel and eventual obliteration of the longitudinal muscle layer and the myenteric nerve plexus. Sclerosing encap-sulating peritonitis is characterized by cocooning of the small bowel by a sheath of new fibrous tissue. The bulk of the encapsulating sheath lies anteriorly with septae dipping between bowel loops to involve the mesentery, which becomes contracted. Histologically there is dense fibrous tissue permeated with a chronic inflammatory infiltrate [1]. Calcification may occur but extensive peritoneal calcification has been consid-ered a separate entity and reported as calcifying peri-tonitis, but some cases have had features of sclerosing encapsulating peritonitis.

The aetiology is usually unknown but is likely to be multifactorial. Early studies implicated chlorhexidine in alcohol sterilizing sprays, acetate dialysate, endo-toxin from in-line bacterial filters, and severe peritonitis [2] with chlorhexidine antiseptic being the only factor identified in a case control study [3]. Heat sterilization of dialysate results in dextrose degradation products which are the major cytotoxicity factor in peritoneal dialysis solutions [4]. Plasticisers and particulate matter in the dialysate as well as the hypertonicity and acidity of the dialysate have also been implicated [4]. Dextrose itself is toxic by passive glycosylation of the submesothelial tissue when the mesothelium is breached due to injury [4].

Clinical features are variable and include abdominal pain, nausea, vomiting, weight loss, loss of ultrafiltration, ascites, and blood-stained dialysate [2]. The onset is often insidious but subacute small-bowel obstruction heralds the onset of severe disease which may progress to complete bowel obstruction with malabsorption and malnutrition. The disease may present first after transfer to haemodialysis. Severe peritonitis may immediately precede the diagnosis but the condition often presents some months after peritoneal dialysis has been stopped because of peritonitis or for other reasons [2]. The diagnosis of severe disease is usually made at laparotomy, but more recently findings on CT scan and ultrasound of the peritoneum have been used to support the diagnosis [5]. Morbidity and mortality from small-bowel obstruction is high, with death due to sepsis and malnutrition [2,6]. Cessation of peritoneal dialysis and transfer to haemodialysis may be beneficial but the disease often persists [2]. Surgical excision of the sclerotic peritoneum in encapsulating disease has generally been unsatisfactory. Resolution of bowel obstruction after renal transplantation has supported treatment with immunosuppression [7].

Sclerosing encapsulating peritonitis in patients on peritoneal dialysis was the subject of several reports from 1980 to 1986 [2,3,6,8]. With the elimination of chlorhexidine sterilizing agent, acetate dialysate, in-line bacterial filters, and a decrease in the peritonitis rate, there should have been a decrease in the incidence of the disease. This does not seem to be the case in Australia.

Subjects and methods

All 40 adult and paediatric dialysis units in Australia were surveyed via questionnaire. Information was sought regarding the number of cases, duration of continuous peritoneal dialysis prior to diagnosis (<3 months interruption), the type of CAPD connector systems, the use of a sterilizing agent, the frequency and type of peritonitis, clinical features, (specifically abdominal pain, blood in dialysate, bowel obstruction, and associated peritonitis); surgical findings, (specifically encapsulated peritoneum, tanned peritoneum, and thickened peritoneum); and the outcome, including the duration of total parenteral nutrition, treatment with immunosuppression, and whether death was secondary to sclerosing peritonitis.

Patients were included if there was either surgical or radiological evidence of sclerosing encapsulating peritonitis or small-bowel obstruction with tanned or thickened peritoneum in the absence of other causes of bowel obstruction.

To determine the number of patients treated by peritoneal dialysis the Australia and New Zealand Dialysis and Transplantation Registry (ANZDATA) was used. A period of peritoneal dialysis was the period of treatment by peritoneal dialysis for each patient. If the patient was off peritoneal dialysis treatment for a continuous period of >90 days and subsequently returned to peritoneal dialysis, this was counted as a new period.

Results

All dialysis units responded. There were 61 patients from 18 units. Seven patients were excluded, leaving 54 patients for analysis. Exclusions were because of persistent peritonitis, two patients; tanned peritoneum with loss of ultrafiltration without evidence of small-bowel destructon, three patients, and perforated bowel, two patients. The mean age was 48 ± 17 years and the female to male ratio was 1.7:1. The mean duration of continuous peritoneal dialysis was 52 ± 30 months with a median of 48 months and range of 8–127 months.

There were 7374 patients treated by peritoneal dialysis from 1978 to 1994 inclusive, giving a prevalence of 0.7%. The mean annual incidence was 2.7 per 1000 periods of continuous peritoneal dialysis with a range of 0–5.5. There were 19 cases diagnosed from 1980 to 1989 and 34 cases diagnosed from 1990 to 1994, giving mean annual incidences of 1.9 and 4.2 respectively for the two periods. The number of periods of continuous peritoneal dialysis >4 years increased from three in 1980 to 34 in 1984 to 117 in 1989 and 146 in 1994 (Figure 1). Sclerosing peritonitis was rare in patients on peritoneal dialysis for less than 2 years, there being only eight cases out of 5661 periods of peritoneal dialysis. The effect of increasing duration of peritoneal dialysis is shown in Figure 2. The prevalence increased from 1.9% for patients on dialysis >2 years to be 6.4, 10.8 and 19.4% in patients on peritoneal dialysis for greater than 5, 6 and 8 years respectively. Only 36 patients had been on continuous treatment for >8 years and 7 had sclerosing peritonitis.

All patients used Baxter CAPD systems and dialysate with lactate as the base. Thirty-two per cent of patients used a spike system, 24% the Leurlock system, 14% the 'O' Set, and 30% Ultraset, Y-Set, or Freeline disconnect systems. Three of the patients using the Leurlock system use chlorhexidine solution to sterilize the ends of the connections. Patients using the 'O' Set sterilized the connector ends in a solution of sodium hypochlorite, and at least two patients stored the lines in the same solution.
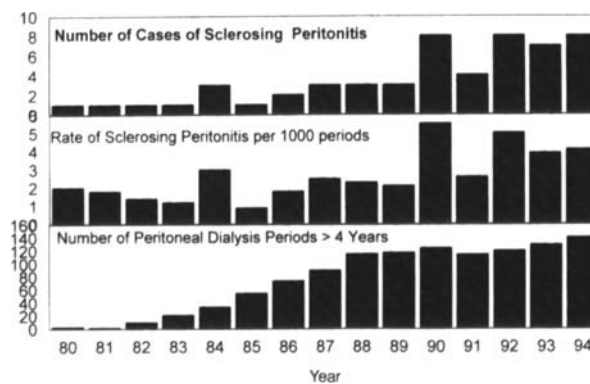

Fig. 1. Sclerosing peritonitis from 1980 to 1994 expressed as the number of cases each year and the rate per 1000 periods of peritoneal dialysis each year and the number of continuous peritoneal dialysis periods >4 years each year.
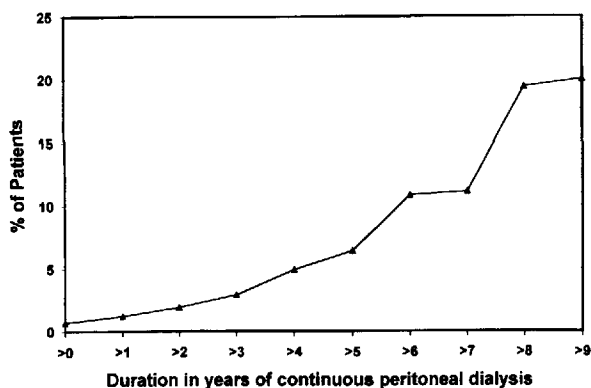
Fig. 2. The prevalence of sclerosing peritonitis in Australia from 1978 to 1994 depending on the duration of continuos peritoneal dialysis.

The mean number of peritonitis episodes was 5 ± 4, or one every 12 patient months. *Staphylococcus aureus* peritonitis occurred in 34% of patients, pseudomonas peritonitis in 11%, and fungal peritonitis in 13%. Two patients have experienced no episodes of peritonitis. One of these patients used chlorhexidine in alcohol as a sterilizing agent with the Leurlock system. Of four patients with duration of CAPD $\leqslant 18$ months, one had fungal peritonitis, one had three episodes of *Staph. aureus* peritonitis, one two episodes of pseudomonas peritonitis with delay of treatment, and one had haemolytic streptococcus and *Enterobacter cloacae* peritonitis.

Sclerosing peritonitis was associated with peritonitis in 40% of cases and with fungal infection in 7%. Typically these patients were asymptomatic or had only minor symptoms until an episode of peritonitis occurred which was resistant to treatment, and then the small bowel would cease to function. Laparotomy revealed evidence of severe sclerosing peritonitis. The bowel function often did not recover and the patient may have died from ongoing sepsis. Haemoperitoneum was reported in four cases or 7%. Small-bowel obstruction was a clinical feature in 40 or 92% of cases. Encapsulated peritoneum was found at surgery in 44 cases and on CT scan in two cases, representing 85% of cases. Pathology of the 'sclerosed peritoneum' revealed keratinization in two cases. Total parenteral nutrition was given in 80% of cases and the duration of treatment was greater than 4 weeks in 42% of cases. Four patients were on home total parenteral nutrition for up to 3 years.

Extensive peritoneal calcification was reported in seven patients. These were sevenof the eight cases who had been on peritoneal dialysis for >7 years. Their mean duration of treatment was 106 months compared to 45 months for the patients without calcification. Five of the cases presented with recurrent small-bowel obstruction, which went onto complete obstruction in one case and bowel infarction in another [12]. Two patients presented with haemoperitoneum. One remained on CAPD for 2 years until she presented with abdominal pain and persistent vomiting, and at laparotomy had a non-functioning bowel. She was

maintained on TPN for 6 weeks, when treatment was withdrawn. The other patient had recurrent postprandial vomiting until she was transplanted 4 months later. Peritonitis developed secondary to a wound infection following removal of the Tenckhoff catheter, leading to unresolved abdominal sepsis and death. At laparotomy loops of bowel were matted together by dense fibrosis.

Treatment with immunosuppression occurred in five patients, two associated with renal transplantation. A female patient aged 27 years developed bowel obstruction in December 1993 after 2 years on CAPD. A diagnosis of sclerosing encapsulating peritonitis was made at surgery. She was transferred to haemodialysis but continued to have recurrent bowel obstruction with five episodes, and twice had surgical excision of the sclerosed peritoneum. Prednisone 30 mg per day was commenced in May 1994. The patient underwent further surgery in June, after which azathioprine 125 mg was added. There was steady improvement with no further bowel obstruction. A successful transplant was performed in December 1994 and there was no further recurrence. A 44-year-old female on CAPD for 9 years presented in October 1992 with recurrent partial small-bowel obstruction which became complete, associated with extensive peritoneal calcification. There was no improvement with transfer to haemodialysis. She was intolerant of any oral intake and was on TPN for 9 months until a renal transplant was performed in April 1994. There was dramatic improvement within 2 weeks and TPN was ceased 12 weeks post-transplant and there has been on recurrence [16]. A 28-year-old male on CAPD for 18 months presented in October 1994 with blood-stained ascites. Laparotomy revealed sclerosing encapsulating peritonitis. Treatment with prednisolone 50 mg and azathioprine 100 mg per day was commenced and the ascites resolved over 6 weeks. A 52-year-old female on CAPD for 28 months developed symptoms of intermittent partial small-bowel obstruction after removal of the peritoneal dialysis catheter. A renal transplant was performed in May 1982, 2 weeks post-transplant she presented with small-bowel obstruction with sclerosing encapsulating peritonitis diagnosed at laparotomy. A 28-year-old female on CAPD for 21 months developed small-bowel obstruction, which improved after removal of the catheter but later relapsed on two occasions. Following surgery and treatment with prednisolone there was slow improvement until she had a transplant 4 months later (Table 1).

Mortality was ascribed to sclerosing peritonitis in 30 patients or 56% of cases. There was no difference in the mortality rate between those with and those without encapsulating peritonitis or those with or those without associated peritonitis. The clinical course was often protracted, with malabsorption secondary to bowel obstruction and sepsis related to ongoing peritonitis or bowel perforation. Withdrawal from dialysis was the ultimate cause of death in at least nine patients or 30% of those who died.

Table 1. Effect of immunosuppression on sclerosing peritonitis

Patient number	Age, sex	Immunosuppression	Transplant	Outcome
1	27, F	Pred 30 mg, later Aza 125 mg	No	Improvement following surgery
2	44, F	CsA, Aza 100 mg, Pred 30 mg	Yes	Dramatic improvement
3	28, M	Pred 50 mg, Aza 100 mg	No	Resolution of ascites
4	28, F	Prednisone	No	Gradual improvement following surgery
5	52, F	CsA, Aza, Pred	Yes	SBO occurred post-transplant

Pred, prednisone; Aza, azathioprine; CsA, cyclosporin A; SBO, small-bowel obstruction.

Discussion

The first reported cases of sclerosing peritonitis in Australia were published in 1981 [8]. The two cases reported were included in this survey, which otherwise relied on clinical recall. The study is limited by being retrospective, with incomplete information on histology, radiology, and clinical features, but is strengthened by the complete national registry of dialysis and transplant patients. There were 54 cases reported from 7374 patients treated by peritoneal dialysis between 1978 and 1994, giving an overall prevalence of 0.7%. Eighty-five per cent of cases met the criteria of sclerosing encapsulating peritonitis, giving a prevalence of 0.6%. This compares with a prevalence of 0.9% for sclerosing encapsulating peritonitis in 6923 CAPD patients in Japan over the same period [9]. The mean annual incidence from 1980 to 1994 was 2.7 per 1000 peritoneal dialysis treatment periods, with a range of 0.9–5.0. The annual incidence in the study from Japan ranged from 0 to 4.3 per 1000 patients undergoing CAPD [9]. In the mid-1980s the annual incidence ranged from 0.9 to 3.0 per 1000 cases at risk, which is similar to that reported for the year 1984 by the EDTA Registry [3]. In Australia, in contrast to Europe, acetate dialysate was not used, nor was chlorhexidine in alcohol spray, although one unit used chlorhexidine to sterilize the ends of Leurlock connectors.

The mean annual incidence increased from 1.9 per 1000 cases at risk for the period 1980–1989 to 4.2 per 1000 cases at risk for the period 1990–1994. With a reduction in the peritonitis rate over this period, one may have expected the incidence to decrease. The factor which did increase was the duration of treatment by peritoneal dialysis. The number of patients on CAPD for >4 years in a particular year increased from 34 or 6% in 1984 to 147 or 12% in 1994 (Figure 1). The median duration of peritoneal dialysis treatment was 48 months for patients developing sclerosing peritonitis. Further evidence that the duration of treatment is a major risk factor for the development of sclerosing peritonitis is the increase in the prevalence of the disease with duration of treatment, reaching 20% after 9 years of continuous dialysis. In the study from Japan there was a marked increased in the number of cases after 1990 and the mean time to developing sclerosing encapsulating peritonitis was

65.4 months, range 10–138 months, which is very similar to this study [9].

With the duration of peritoneal dialysis being so important other factors apart from peritonitis must be considered. Glucose-based dialysate may be an important factor. Hypertonic glucose can cause diabetiform-like changes in the subserosal tissue. Dextrose degradation products form as a consequence of heat sterilization of glucose-based dialysate and are toxic to the peritoneum. The levels are monitored during commercial production; however, levels rise with ageing of the dialysate and is accelerated by storage at high temperature. Sterilization by filtration avoids the production of the dextrose degradation products but it is too expensive for commercial use. Plasticisers and particulate matter in the dialysate as well as the hypertonicity and acidity of the dialysate have also been implicated [4].

Peritonitis, however, appears critical in the development of sclerosing peritonitis in the vast majority of cases. With bacterial or chemical peritonitis there is loss of the mesothelium of the peritoneum and if there is failure of remesothelialization, the subserosal stroma is exposed to high concentrations of glucose. There is a thickening and reduplication of basement membranes, and changes in the collagen and matrix thought to be secondary to passive glycosylation. The changes are also compatible with chronic exposure to bacterial toxins presumably derived from the catheter biofilm [1]. With peritonitis there may be an imbalance between fibrinolysis and fibrinogenesis. The mesothelium has the capacity to secrete prostacyclin, lubricant surfactant, and tissue plasminogen. With loss or damage to the mesothelium there is decreased fibrinolysis at a time of increased fibrinous exudate. A fibrin mantle forms that is progressively organized, resulting in a sheath of new fibrous tissue. The uncontrolled proliferation of mesochymal stem cells may be a key factor in the production of excess fibroconnective tissue [1].

Peritoneal dialysis acts to remove the fibrin and inflammatory mediators, so cessation of dialysis may promote fibrin accretion and has been implicated in the progression of sclerosing peritonitis. In the current study 40% of cases had associated peritonitis at the time of diagnosis. It is thought that particularly virulent organisms such as sensitive *Staph. aureus*, pseudomonas, and fungi may be more likely to cause the disease and may lead to the early onset of peritoneal

sclerosis [2]. The diagnosis of sclerosing encapsulating peritonitis may be problematic when it follows severe infection, especially in cases of faecal or fungal peritonitis, as the severe infection may lead to a pseudomembrane, but not usually to encapsulation. Also, severe persistent infection may lead to a prolonged ileus. Four patients were excluded because of this difficulty. In the current study 34% of patients experienced a *Staph. aureus* infection, 11% pseudomonas, and 13% fungal, which appears to be excessive, and the four patients with duration of CAPD $\leqslant 18$ months all had severe and/or recurrent peritonitis. The overall peritonitis rate however did not appear to be excessive with a mean number of five episodes per patient with a mean duration of CAPD of 57 months, or a peritonitis rate of one every 12 patient months. One may have predicted this as patients with frequent peritonitis do not remain on CAPD for long periods.

Two patients did not experience peritonitis. One used chlorhexidine in alcohol solution to sterilise the ends of a Leurlock connecting system. In a review of the literature in 1986 [6] three cases did not experience peritonitis. All were on intermittent peritoneal dialysis where formaldehyde disinfectant was most probably used. However, in the study from Japan, five cases never experienced clinical peritonitis and were not exposed to intraperitoneal antiseptics [9].

Extensive peritoneal calcification was not a feature of the early reports of sclerosing encapsulating peritonitis. It has generally been reported as 'calcifying peritonitis' with 9 possible cases reported in the literature [10–16]. These cases are characterized by eggshell calcification of the visceral peritoneum outlining the small bowel, plus parietal peritoneal calcification. Pathological specimens show loss of mesothelium, extensive fibrosis, and calcification with a sparse inflammatory infiltrate. The clinical features are a long duration of CAPD, haemoperitoneum, abdominal pain. and less frequently partial small-bowel obstruction and loss of ultrafiltration. There is a good clinical outcome on transfer to haemodialysis. However one case, maintained on CAPD, included in this study, went onto develop small- and large-bowel infarction associated with unresolved peritonitis and a extension of the calcification including involvement of the bowel wall [12]. Another case, also included in this study, remained on CAPD for 2 years until she presented with persistent vomiting and had a non-functioning bowel which did not improve [11]. In this study there were seven patients with extensive calcification. The two mentioned above, four who had features characteristic of sclerosing encapsulating peritonitis, including one who went on to complete bowel obstruction [16], and one who developed a non-functioning bowel associated with intra-abdominal sepsis. All had been on peritoneal dialysis >8 years. Hyperparathyroidism has been implicated n the development of peritoneal calcification, as has local calciphylaxis.

Calcifying peritonitis can be considered part of the spectrum of sclerosing peritonitis and there may be features of encapsulating peritonitis. If peritonitis develops in these cases the outcome is poor. It would appear prudent to perform an abdominal X-ray or CT scan on all patients who had been on CAPD for >5 years, and if extensive peritoneal calcification and/or features of encapsulating peritonitis are present, to transfer the patient to haemodialysis.

Treatment has invariably meant transfer to haemodialysis although cessation of peritoneal dialysis may accelerate the sclerosing process [2]. Some patients recover bowel function with time but home parenteral hyperalimentation may be required [6,16]. Total parenteral nutrition is critical, as malnutrition predisposes the patient to sepsis and other complications. Total parenteral nutrition was used in 82% of cases in the current study. One patient was maintained on home parenteral nutrition for 3 years.

Surgery is usually difficult and hazardous, with the risk of bowel perforation and recurrent sepsis. If there is bowel necrosis, primary anastomosis should not be attempted [17]. In some cases the thickened membrane has been readily stripped off the bowel, revealing a relatively normal small-bowel serosa [8]. Recurrence after surgery does occur, which may be reduced by concomitant immunosuppression. Immunosuppression has been recommended prior to surgery to reduce the adhesion of the sclerosed peritoneum to the bowel wall [19].

Junor and McMillan [7] were the first to report better outcomes in patients with sclerosing encapsulating peritonitis who received immunosuppression. In their study of 17 cases which were mainly secondary to chlorhexidine, four patients were transplanted using prednisolone 20 mg daily and azathioprine or cyclosporin, and a further patient transferred to haemodialysis received prednisone 10 mg and azathioprine 50 mg daily. Of the five immunosuppressed patients none died from sclerosing peritonitis, while the 12 who did not receive immunosuppression died with recurrent bowel obstruction within 1 year [7].

In the current series one patient had a dramatic improvement in bowel function following renal transplantation, and this case has been reported previously [16]. In contrast, another patient had progression of the disease despite renal transplantation. Presentation after successful renal transplantation has been reported. All three patients had successful lysis of the fibrotic tissue without recurrence, which may have been due to their immunosuppressive therapy [18]. Bhandari *et al.* [19] reported a case in which after 6 weeks of treatment with azathioprine and corticosteroids there was a marked improvement in the degree of adhesion. In a recent review of their experience with sclerosing peritonitis Selgas *et al.* [20] noted an apparent beneficial effect of renal transplantation and/or immunosuppression, including intraperitoneal corticosteroids.

In conclusion, in this retrospective study, the prevalence of sclerosing peritonitis is quite low at 0.7%, but greatly increased with the duration of CAPD to >10% after 6 years. Severe peritonitis may lead to the early onset of sclerosis, while patients with sclerosing peri-

tonitis who develop peritonitis have a poor prognosis. Extensive peritoneal calcification can be considered part of the spectrum of sclerosing peritonitis, and is generally seen after several years of CAPD. The morbidity and mortality of sclerosing peritonitis are high and a trial of immunosuppression should be considered. It would appear prudent to screen for sclerosing peritonitis in patients on CAPD for >5 years.

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